Nephrology

Version 5.2

Corrected, Updated, Lighter

PLAB 1 Keys is for PLAB-1 and UKMLA-AKT (Based on the New MLA Content-Map)

With the Most Recent Recalls and the UK Guidelines

ATTENTION: This file will be updated online on our website frequently!

(example: Version 2.1 is more recent than Version 2, and so on)

Key 1	**Rhabdomyolysis**

• As skeletal muscles are dying → they release (**Myoglobulin**, **Potassium**, **Creatine Kinase**…).

• **Common Scenarios and Hints: (Important √)**

√ A person was trapped for several hours under a heavy object.

√ A fall followed by a long period of lying on the floor.

√ An elderly with frequent falls presents with Acute kidney injury.

√ IV drug abuser was found on the floor not moving for a long period.

√ Long-distance run (eg, Marathon runner) "Severe Exertion/ Severe Dehydration".

√ Severe Crush injury.

√ Exercise-induced rhabdomyolysis (e.g. in athletes)

± Hematuria (**Reddish Brown** or **Tea-coloured urine**) (False Positive as the cause of redness is myoglobulin (which has heme), while RBCs are not found in urine dipstick).

± Hypotension.

± AKI "Acute kidney injury" → (High urea and Creatinine).

± Very high CK (Creatine Kinase).

√ Although uncommon, one of the side effects of **statins** is Rhabdomyolysis. √

• **Myoglobulin** is **nephrotoxic** and thus can lead to **Acute Kidney Injury (AKI).** Therefore, rehydration with **IV fluid** is an **essential initial step**. That's why Rhabdomyolysis is a medical emergency that you have to be aware of!

• **ECG** must be performed as the released potassium from the dying muscles (**hyperkalemia**) can be dangerous. If ECG changes suggesting hyperkalemia (**Tall tented T wave, Wide QRS**) are found:

→ Protect the heart by giving **IV Calcium Chloride** or IV **Calcium Gluconate before anything else!**

◙ Important points on Rhabdomyolysis:

√ Main Complications of Rhabdomyolysis → **AKI** and **Hyperkalemia**.

√ Initial management → **IV fluid** (to try to avoid acute kidney injury).

√ Initial Investigation for management → **ECG**

√ If **Tall T wave, Wide QRS**, the initial line → give **IV calcium chloride/ gluconate**.

√ The best **initial** test that is **specific for Rhabdomyolysis** → **Urine analysis** → Reddish-brown (Tea-coloured) → Falsely +ve hematuria.

√ **To confirm** → **CPK level** (**Creatine Phosphokinase**) "it indicates muscle necrosis".

√ Other lines of treatment include: Sodium Bicarbonate ▌Dialysis (in severe cases)

Elaboration

Example **(1),**

A man has just finished a long-distance run and presents with myalgia, red-brown urine. Urine dipstick is positive for blood but without RBCs on microscopy. ECG shows Tall T waves, Widened QRS and small P waves.

• The initial step in management → **IV Calcium chloride/ Gluconate**

It is obvious he has **Rhabdomyolysis**.

As there are ECG changes suggesting **Hyperkalemia**, we should protect the heart before anything else by giving IV Calcium. **First things First**!

Otherwise, **IV fluid** is the initial step.

Example **(2),**

24 YO mountain climber was rescued after being trapped under a heavy rock for about 10 hours. He has dark urine with impaired creatinine and urea. His SBP is 100 mmHg and HR is 125 bpm.

- The most appropriate management → **IV fluid normal saline**.
- The likely cause of his renal failure → **Myoglobin "reno-toxic"**

√ He has **Rhabdomyolysis (**Skeletal muscle breakdown due to crush injury or prolonged immobilised muscles**).**

√ As the question did not mention ECG changes of hyperkalemia (e.g. Tall T wave), the initial step would be **IV fluid rehydration** to try to avoid further renal damage.

√ **Myoglobin** is **Nephrotoxic**.

Example **(3),**

A 22 YO Known HIV Positive male was found comatose on the floor by his friend. He was not seen for 2 days. On the ED, the patient is confused and

disoriented. His labs reveal serum Urea level of 47 mmol (normal: 2-7) and Creatinine 1070 (Normal: 70-150). Sodium is normal, Potassium is 5.6.

The **best** investigation to perform → **Creatine Kinase**.

√ This is most likely a case of **Rhabdomyolysis**.

√ Prolonged immobilisation (e.g. in coma) → Muscle Ischemia → Release of myoglobin, Creatine Kinase, Potassium and others → Acute Kidney Injury (as seen here).

√ To confirm → **Serum CPK** "**Creatine PhosphoKinase**".

√ Note, if the Potassium is ↑ and the "ECG" is given as an option, pick it especially if the question asks about the "**initial**" step.

Example **(4),**

A 60 YO man known case of hypertension, diabetes mellitus and a previous TIA presents complaining of a 5 day of diffuse muscle pain and weakness in his lower limbs. He is on: ramipril, bisoprolol, aspirin, metformin and simvastatin. His urine shows myoglobin. His kidney function tests are deteriorated. His serum creatinine kinase (CK) is 3000 (Normal: 45-260).

√ The likely Dx → Rhabdomyolysis.

√ The likely causing medication that needs to be stopped → Simvastatin.

Although uncommon, one of the side effects of **statins** is **Rhabdomyolysis**.

***Example* (5),**

A man had a wardrobe over him for a long unknow period. He was taken to the ER. He is confused and disoriented. What is the most appropriate investigation?

→ **Creatine Kinase**.

Key 2

Goodpasture Syndrome

Acute rapidly progressive glomerulonephritis [+] Pulmonary alveolar hemorrhage

Expressed as:

Hematuria (Kidney involvement) [+] **Hemoptysis (Lung involvement)**

[+] **Impaired KFTs**, obviously.

◙ The most **appropriate** "**initial**" investigation

→ **Anti-glomerular basement membrane antibodies** (Anti-GBM antibodies)

◙ The most "**accurate**" investigation

→ **Lung or Kidney Biopsy** "Crescentic Glomerulonephritis"

• Chest X-ray → Patchy interstitial infiltration (Intra-pulmonary bleeding).

Important Notes:

Goodpasture Syndrome (GPS)	Hemoptysis + Hematuria (Lung + Kidney only) (Abnormal Urea and Creatinine)	**Anti-Glomerular Basement Membrane Antibodie.**
Alport Syndrome (X-linked)	**GPS (Hemoptysis + Hematuria) + Cannot see** **+ Cannot hear (SNHL)**	
Alpha-Antitrypsin deficiency	Hemoptysis + Jaundice (Liver)	
Churg Strauss (Eosinophilic Granulomatosis with Polyangiitis)	Asthma, Eosinophilia + Other organs.	p-ANCA

	Wegener's Granulomatosis (Granulomatosis with Polyangiitis)	Upper Respiratory problems (Sinusitis/ Nasal septum perforation/ Epistaxis) + Hematuria.	c-ANCA
	Hemolytic-Uremic Syndrome (HUS)	Diarrhea that turns to bloody diarrhea + Hematuria (AKI)	

Key 3

Careful!

The sentence "**itching after a hot shower**" does not always mean Polycythemia rubra vera (PRV)!

◙ **Itching (worse after hot bath)** + Pale skin + Peripheral Oedema + ↑ Skin Pigmentation + (lethargy, tiredness…)

→ **Chronic Renal Failure**

√ ***Itching*** ► due to ↑ serum urea "**Uremia**" (seen in late stage renal failure).

√ ***Pale***, ***Tiredness*** ► due to ↓ Erythropoietin and thus Anemia.

	√ ***Peripheral edema*** and ***hyperpigmentation*** are also seen in CKD. √ Note that in **liver failure** → Ascites, Jaundice, bleeding. ◙ **Itching (worse after hot bath)** ± Red skin "Flushed/ Plethora due to ↑ Hb" ± Splenomegaly ± Burning sensation in fingers and toes ± Gout + High Hb → **Polycythemia Rubra Vera**. ◙ **Itching (worse after hot bath)** + Linear tracks on skin (Burrows) → **Scabies**. ♦ PRV → Red ▌♦ Liver Failure → Yellow "Jaundice" ▌♦ CRF → Pale
Key 4	◙ **Hyponatremia** can occur after **inappropriate IV fluid** therapy such as by using 5% dextrose. This is called (**Iatrogenic cause**). ◙ In sepsis, the allowed IV fluid is either Ringer Lactate or NS (0.5% NaCl), these won't cause dilutional hyponatremia. ◙ Hyponatremia after inadequate IV fluid treatment could be **dilutional**. ***Example,*** A patient with abdominal sepsis (e.g. biliary sepsis) was treated with IV antibiotics and IV fluids. Later on, he was found to be hyponatremic (127 mmol/L) with normal Potassium and Normal kidney function tests (Urea and Creatinine).

	The likely cause → **Iatrogenic**. *Normal Serum Na^+ → 135-145 mmol/L*
Key 5	◙ Massive **hemorrhage** (e.g., during surgery) and **hypotensive shock** + High creatinine → **Acute Tubular Necrosis** (**ATN**) (the commonest renal cause of acute kidney injury). √ "The kidneys need to remain well hydrated/ perfused to avoid acute "**tubular**" necrosis". √ "Prolonged **ischemia → "low perfusion"** to kidneys → **Dying tubules** = **necrosis** = **Acute tubular necrosis** → AKI" --- **Acute Interstitial Nephritis** example: **Allergy** (e.g., **drug intake** followed by **rash**, **fever**) + **Hematuria** + **↑ creatinine**.
Key 6	√ **25**-alpha-hydroxylation of vit. D occurs in → **Liver**. √ **1**-alpha-hydroxylation of vit. D occurs in → **Kidney**. 25-hydroxyvitamin D is formed in liver → Then, an enzyme called **(1-alpha-hydroxylase**) in **kidneys** converts it to the active form, which is

→ 1,25-dihydroxyvitamin D.

So, why is there vitamin D deficiency in chronic renal failure?

→ **due to reduced activity of 1-alpha-hydroxylation**.

Mnemonic

(**K**) before (**L**) in alphabetics.

(**1**) before (**25**)

Kidney → **1** ▌**L**iver → (**25**)

Another mnemonic

1K

1=Kid

Key 7

Chronic and Acute Pyelonephritis

When you see **Hypertension** + Hx of **repeated urinary tract infections** (**UTIs**)

→ Think of: **CHRONIC Pyelonephritis**

Repeated UTIs → Renal Scarring → Chronic Pyelonephritis → Hypertension

When you see LOIN/BACK pain + Features of UTI → Acute Pyelonephritis.

Do not get fooled by the patient being pregnant. Remember, **Pre-eclampsia** occurs **after the 20th week** of gestation + **Hypertension** + **Proteinuria**.

Caution!

Repeated UTI **but** suddenly developed (**loin or back pain**, **fever**, **rigors**), [±] Urinalysis shows blood, nitrites, leucocyte esterase.

→ **ACUTE Pyelonephritis**.

- **Chronic** pyelonephritis has no active infection! The symptoms may be 2ry to renal failure.
- **Acute** Pyelonephritis: See below ↓

A pregnant woman presents with fever, rigors, lower abdominal pain, dysuria and frequency. The pain was at the lower abdomen then it becomes more generalised and radiates to the right loin.

→ **Acute Pyelonephritis**.

• Pyelonephritis = inflammation of the **pelvis** of the kidney (usually due to **Ascending UTI**).

• Some RFx → Pregnancy, Stones, Vesico-ureteric reflux (VUR), DM.

• Suspect it if

√ **Lower UTI features** (**dysuria** -burning or difficult urination, frequency, urgency, suprapubic (lower abdominal) pain]

√ [+] **Fever**, Rigors, **Loin pain** or **Back pain**

√ [±] Urinalysis → blood, **nitrites**, leucocyte esterase.

IN SHORT,

√ **Dysuria**, **frequency**, **urgency ± lower abdominal Pain** → Lower UTI

→ *Trimethoprim* (**or**) *Nitrofurantoin*

√ **The above** + **Loin** or **Back** pain + **fever** → Acute pyelonephritis (Upper UTI)

→ *Ciprofloxacin* (**or**) *Co-amoxiclav*.

All these medications are important to memorise!

◙ Investigations:

• Do **Urinalysis** → to look for (blood, protein, nitrate, leucocyte esterase).

• Do **Urine Culture and Sensitivity** before commencing empirical antibiotics.

√ (midstream specimen for adult and old children)

√ (sometimes in young children → clean catch or via catheter or even via suprapubic aspiration).

Important, in Acute pyelonephritis, **once the urine sample for culture and sensitivity has been sent** → **Start empirical Antibiotics immediately**.

The empirical antibiotics may need to be changed after the culture results.

♦ **Important**,

the most common causative organism of **UTI** → **E. coli** (Gram -ve).

◙ **Treatment** (often requires admission)

Antibiotic lines differ from hospital to another; however, the following regimens are advised by NICE:

• **Non-pregnant women** • **Men** • Patients with **indwelling catheters**	√ **Ciprofloxacin**: 500 mg BID for 7 days. [**or**]: √ **Co-amoxiclav** 625 mg TID for 14 days. BID: Twice a day ▌TID: 3 times a day.
Children	√ 1st line → **Co-amoxiclav**. √ 2nd line → **Cefixime**.

Pregnant ♀ who **does not require admission**	**Cefalexin (PO)** 500 mg BID for 10-14 days. "important √"

If the patient is admitted, these antibiotics are given Intravenously "IV".

In short:

- **Acute pyelonephritis → Urine analysis + Urine Culture → Then Start Antibiotics.**
- **Lower UTI** → Trimethoprim (**or**) Nitrofurantoin.
- **Upper UTI** → Ciprofloxacin (**or**) Co-amoxiclav.
- **Pregnant** → Cefalexin.

Quick info:

√ **Small Kidneys** + **Hypertension**

Think → **Bilateral renal artery stenosis** **[OR]** **Chronic Pyelonephritis**.

√ **ACE inhibitors** are **contraindicated** in **Bilateral Renal artery stenosis**.

(see Vascular chapter).

Key 8	• It is sometimes **normal** to find mild **proteinuria** on urine dipstick (traces up to +1) especially in those who regularly **exercise** such as **gym attendees**. • If no symptoms and they are healthy regardless of the mild proteinuria → **Repeat the test**. • **If still high** → Do ***24-hour urine specimen test*** or ***Protein Creatinine Ratio***. • Never refer a patient with an "isolated proteinuria" to a Renal Clinic. As a GP, you need to investigate further.
Key 9	**Nephrotic Syndrome** ◙ **Triad of:** 1. **Proteinuria** (> 3g/24hr). 2. **Hypoalbuminaemia** (< 30g/L) and 3. **Oedema** ◙ In **children**, the peak incidence is **between 2 and 5 years** of age.

◙ Around 80% of cases in children are due to **Minimal change glomerulonephritis**.

*Note that **microscopic hematuria** can be seen in 10-30% in minimal change disease.*

◙ **Minimal change disease** almost always presents as **nephrotic syndrome**, accounting for 75% of cases in children and 25% in adults.

◙ The condition generally carries a **good prognosis** with around 90% of cases responding to **high-dose oral steroids**.

◙ Other features include **hyperlipidaemia**, **hypercoagulable state** (due to loss of antithrombin III) and **liability to infections** (due to loss of immunoglobulins).

◙ Features of minimal Change glomerulonephritis:

√ **Nephrotic syndrome**.

√ **Normotension** – hypertension is rare.

√ **Selective proteinuria** (only intermediate-sized proteins such as ***albumin*** and ***transferrin*** can leak through the glomeruli).

√ **Renal biopsy** (The Definitive Diagnostic Test)

→ **Electron microscopy shows fusion of podocytes.**

Example (1),

A 5 YO child presents with ↑ weight, Puffy eyes, Lower limb swelling. He is otherwise healthy and happy. Urine Dipstick reveals +3 Proteinuria.

- What is the **next** best step?

We suspect Nephrotic Syndrome. 2 of the triad are given; **Proteinuria** and **Oedema** (puffy eyes, swelling, increase weight). So, to diagnose it, request

→ **Serum albumin levels**. (**to complete the aforementioned Triad**).

Another valid answer → → **Urine protein to creatinine ratio**.

Once diagnosed, we can refer to renal clinics.

- What is the **best diagnostic** test?

→ **Renal Biopsy**. (**Electron microscopy → fusion of podocytes**).

Example (2),

2 YO child was noticed by his mother to be gaining weight regardless of his poor feeding. There is gradual swelling of his face, feet and legs. The child feels fatigued, and his urine is noted to be *foamy*.

The likely Dx → **Nephrotic Syndrome**.

The most appropriate Investigation → **24-Hours urinary protein**.

Another valid answer → → **Urine protein to creatinine ratio**.

Remember, Nephrotic Syndrome = **Proteinuria** + **Albuminemia** + **Oedema**.

Thus, **24-hour protein in urine** + **serum albumin** levels are required to help in diagnosis in this case as he already had oedema manifested.

• Note, one important explanation of **foamy urine** is the presence of **high protein** in it.

Example (3),

40 YO ♂ known hypertensive presents with tiredness, lethargy, fluid retention and proteinuria. His serum albumin is found to be low.

The most "**Definitive**" diagnostic test → **Renal Biopsy** (it shows the etiology).

• This patient has **proteinuria**, **albuminemia** and **fluid retention (swelling)** → the triad of **Nephrotic Syndrome**.

• In the exam, always **pay attention to the question words**. It asks about "the **definitive diagnostic** test". In Nephrotic syndrome, Kidney Biopsy is definitive as it can show the exact etiology.

Example (4),

6 YO child presents with progressively increasing swelling of his face, scrotum and legs. His urine is frothy. Renal biopsy shows no abnormalities on light microscopy. However, electron microscopy reveals abnormal fused podocytes.

The likely Dx → **Minimal change disease**.

Example (5),

5 YO child presented a week ago with progressively increasing swelling of his face, scrotum and legs and with frothy urine. Renal biopsy confirmed the Dx of Nephrotic syndrome and accordingly the patient was started on steroids. He presents now with flank pain, hematuria and fluctuating urea levels. A diagnosis of renal vein thrombosis is made. What is the most likely cause for this renal vein thrombosis?

→ **Antithrombin III deficiency**

√ Remember that **proteinuria** is one of the triad of Nephrotic Syndrome.

√ Proteinuria means **loss of proteins in urine** such as **albumin** and **anti-coagulation factors** including **Anti-thrombin III**.

	√ Loss of Antithrombin III → ↑ Coagulation (↑ Thrombosis). √ Rx → Anti-coagulation.
Key 10	• Prolonged **vomiting** and **diarrhea** can lead to → **Dehydration**, **Hypokalemia** (↓ K). • **Dehydration "Hypovolemia" can lead to → Acute Kidney Injury (AKI).** • Prerenal causes "Especially hypovolemia" is the most common cause of AKI. • Once there is **AKI** → **↓ eGFR** → Kidneys are unable to well execrate Potassium, Creatinine, Urea…etc. → **Hyperkalemia**, **↑ serum urea** and **Creatinine**.
Key 11	In CT scan with **contrast**, to reduce the risk of "**Contrast Induced Nephropathy**" → **Drink Plenty of Fluids**. Another correct answer is → **IV fluid normal saline (0.9% NaCl)** pre and post procedure especially in high risk patients such as elderly with DM. • Additional Note, **metformin** is **Nephron-harmful** and thus needs to be ceased "**stopped**" before any **contrast** study.

Key 12

The steps (approach) to determine the type of the blood gas abnormality.

1. Is the patient **acidaemic** (**pH <7.35**) or **alkalaemic** (**pH >7.45**)?
2. Respiratory component: What has happened to the **$PaCO_2$**?

- **$PaCO_2$ > 6.0** kPa suggests a **respiratory acidosis** (or respiratory compensation for a metabolic alkalosis)
- **$PaCO_2$ < 4.7** kPa suggests a **respiratory alkalosis** (or respiratory compensation for a metabolic acidosis)

3. Metabolic component: What is the **bicarbonate** level/base excess?

- **bicarbonate < 22** mmol/l (or a **base excess < - 2mmol**/l) suggests a **metabolic acidosis** (or renal compensation for a respiratory alkalosis)
- **bicarbonate > 26** mmol/l (**or a base excess > + 2mmol**/l) suggests a **metabolic alkalosis** (or renal compensation for a respiratory acidosis)

Simply, know that CO2 is an Acid, and Bicarbonate (HCO3) is an Alkali.

Example (1):

pH 7.17 (Normal: 7.35-7.45)

PCO2 2.5 (Normal: 4.7-6 kPa)

Base excess -14 (Normal -2 to +2)

→ **Metabolic acidosis** (with partial respiratory compensation).

♦ **As the pH < 7.35** → definitely Acidosis.

♦ **PCO2 (the acid) is low** → this is compensation by the lungs; they try to breathe quickly to get rid of the CO2 (the acid) to buffer the acidity. The patient might present with tachypnea or SOB.

♦ **Base excess is very low** → metabolic acidosis.

Example (2):

An elderly man was found on the floor unconscious by his neighbours. The ambulance crew came. His Systolic BP was 65mmHg. He was resuscitated in the ambulances (given 1.5 L NaCl 0.9%). He was further resuscitated in the emergency department. He mentions that he had severe diarrhea over the last 2 days. His labs show:

pH 7.18 ▌Base excess -13 ▌Lactic acid 6 (high)

Urea and Creatinine are high ▌CRP 160 (high)

His blood gas interpretation → **Metabolic Acidosis**

As his pH < 7.35 → Acidosis

His Base excess is very low (< -2) → Metabolic Acidosis.

This patient had profuse diarrhea for 2 days. Remember that profuse diarrhea can lead to loss of HCO3 "Bicarbonate" and thus metabolic acidosis.

Also, remember that profuse diarrhea can lead to → Hypovolemia "Dehydration", which is an important prerenal cause for AKI. That's why his renal functions are impaired.

Also, once AKI develops → ↓ eGFR → accumulation of K+ and others. (Hyperkalemia).

Key 13

Managing Renal Stones (also in urology section)

Generally, the Stone Size Rule:

◙ If stone size **< 0.5 cm** (< 5 mm) → ↑ fluid intake to get rid of it in urine.

◙ If stone size **0.5 cm – 2 cm** → two options:

√ ESWL (Extracorporeal Shock wave Lithotripsy) "**preferred**", or:

√ Ureteroscopy with dormia basket.

◙ If stone size **> 2 cm** → Percutaneous Nephrolithotomy.

HOWEVER!

◙ If the patient has only **one functioning kidney** (e.g. Hx of the removal of one kidney) and has a stone (**OF ANY SIZE**) with dilatation of the pelvicalyceal system (**PCS**) ± Anuria, Fever [**Obstructive Uropathy**] →

The **INITIAL** thing to do is to **decompress** the **PCS** to save the remaining kidney. This is done by → **Percutaneous Nephrostomy**

◙ Similarly, even if the patient has **2 kidneys**, if he develops **AKI** (impaired urea and creatinine), **fever** and **Hydronephrosis** (these together with the presence of **stones** are indicative of **Obstructive Uropathy**), we shall go for (**Percutaneous Nephrostomy**) in order to temporarily and instantly decompress the renal collecting system regardless of the stone size!

Note, Percutaneous nephrostomy is different form Percutaneous nephrolithotomy.

• **Percutaneous Nephrostomy** → stoma "catheter" to the Pelvicalyceal system of the kidney for decompression (Draining the obstructed fluid in kidney).

• **Percutaneous Nephrolithotomy** → removal of urinary stone percutaneously via a scope (if size > 2 cm).

Example (1),

46 YO ♂ with Hx of left nephrectomy 10 days ago presents with fever, inability to pass urine for the last 20 hours. **Ultrasound reveals** an 8 mm stone in the

left lower ureter with dilatation of the pelvicalyceal system. What is the best **INITIAL** step in management?

• The best initial step → **Percutaneous Nephrostomy**.

The (Stone Size Rule) does not apply here. This patient has **obstructive uropathy** with impending renal failure. We need to, initially, **save his remaining kidney by decompressing the fluid retention in the PCS**. This can be done by → Percutaneous **Nephrostomy**. Afterwards, we can manage the stone based on the stone size role; (ESWL) in this case.

In summary:

◙ Loin pain + Stone ± Hydronephrosis → Manage according to the **stone size**.

◙ Loin pain + Stone + **Hydronephrosis** [+] **AKI** [+] **Fever**

→ **Percutaneous Nephrostomy**.

Example (2),

36 YO ♂ presents with severe loin pain, nausea and vomiting. Ultrasound shows right hydronephrosis. Non-enhancing CT reveals a 3.1 cm stone at the level of the minor calyx. What is the most appropriate management?

• The most appropriate management → **Percutaneous Nephrolithotomy**.

	He has **2** working kidneys with **no** AKI. According to the "**Stone Size Rule**", **stone > 2 cm** → **Percutaneous Nephrolithotomy**. If there was AKI with fever, the "initial" step would be to decompress the PCS by percutaneous nephrostomy. *We hope the concept is clear.*
Key 14	A **Hypertensive patient** with **CKD** "Chronic Kidney Disease" with GFR is still > 30 and ACR is > 30. → **Give ACE inhibitors (e.g., ramipril, lisinopril)** to manage HTN or ARBs. [**They slow the progression of CKD**]. **However, avoid ACEi and ARBs if GFR < 30 or ACR < 30 in patients with HTN.** (**ACEi** (eg, ramipril, lisinopril) or **ARBs** (eg, losartan) are the preferred drug for Hypertensive patients of any age/ ethnicity with **chronic kidney disease** as long as **GFR > 30** and Albumin: Creatinine Ratio **(ACR) > 30** ◙ Also, remember: **DM type 2 + HTN** → always consider **ACEi** (it is reno-protective) unless severe renal impairment (GFR < 30).

	◙ **ACEi and ARBs can be used to delay the progression of end stage renal failure. They can be used in the following situations (as long as):** • ACR ≥ 70 (ACR = urinary Albumin: Creatinine ratio). • ACR ≥ **30** (If there is **HTN**). • ACR ≥ **3** (If there is **DM**). ◙ **Important notes:** • In addition to ACE inhibitors → **Sodium-Glucose Co-Transporter-2** (**SGLT2**) **inhibitors** provide **renal**, **cardio**, and **diabetic** protection. • Thus, you can add it to ACE inhibitor if needed (eg, if HbA1c is still > 48 mmol/mol). • SGLT2 inhibitors are beneficial in patients with Type 2 diabetes + **cardiovascular disease**/ **risk**. • Examples of **SGLT2 inhibitors** → **flozin** → Dapagliflozin, Canagliflozin, Empagliflozin.
Key 15	◙ The most common cause of **Nephrotic Syndrome** in **Children** → **Minimal Change Glomerulonephritis**. ◙ The most common cause of **Nephrotic Syndrome** in **Adults**:

	√ in Caucasian ▌White people ▌or if unspecified ethnicity → **Membranous GN**. √ in Africans ▌Black ▌Americans ▌Hispanics → **Focal Segmental GN**. Generally, for PLAB 1, the most common cause of **Nephrotic Syndrome** in **Adults** (especially > 40 YO) → **Membranous GN**. --- **Membranous glomerulonephritis** • Presentation: proteinuria / nephrotic syndrome / chronic kidney disease • Cause: idiopathic (Mainly), infections, rheumatoid drugs, malignancy • **Prognosis**: ♦ 1/3 of the cases → Remission. ♦ 1/3 of the cases → Partial Remission. ♦ 1/3 of the patients → progress into End-stage Renal Failure.
Key 16	**Types of Glomerulonephritis** "For Reading" There are 2 Main types 1) Presents with Nephritic syndrome: eg, rapidly progressive (Goodpasture), IgA 2) Presents with Nephrotic Syndrome: eg, minimal change, membranous, focal seg.

◙ **Typically presents with Nephritic Syndrome (haematuria, hypertension):**

♣ **Rapidly progressive glomerulonephritis (Crescentic glomerulonephritis):**

√ Rapid onset, often presenting as acute kidney injury.

√ Causes include Goodpasture's (hematuria + Hemoptysis), ANCA positive vasculitis.

♣ **IgA nephropathy (Berger's disease) and Mesangioproliferative GN:**

√ Typically, young adult with haematuria **1-2 days** after a URTI or less often after gastroenteritis.

◙ **Typically presents with Nephrotic Syndrome (proteinuria, oedema):**

♣ **Minimal change disease:**

√ Typically, a child with nephrotic syndrome (Edema, proteinuria, albuminemia)

• Minimal change disease accounts for 80% of Nephrotic Syndrome cases in children.

√ Causes: Idiopathic (mainly), Hodgkin's, NSAIDs

√ Good response to steroids

√ Renal biopsy on electron microscopy → Fusion of Podocytes.

♣ **Membranous glomerulonephritis:**

√ Presentation: proteinuria / nephrotic syndrome / chronic kidney disease.

√ Cause: idiopathic (mainly), infections, rheumatoid drugs, malignancy.

	√ 1/3 resolve, 1/3 respond to cytotoxics, 1/3 develop chronic kidney disease. ♣ **Focal segmental glomerulosclerosis** √ May be idiopathic or secondary to HIV, heroin. √ presentation: proteinuria / nephrotic syndrome / chronic kidney disease.
Key 17	◙ Suspect CKD "Chronic Kidney Disease" if: ***Anemia*** + ***Hypocalcemia*** and ***Hyperphosphatemia*** + ***Small Kidneys*** on U/S (< 9 mm). ◙ Patients with late **CKD** usually have **Hypocalcemia** which results in ***tingling***, ***numbness***, ***paraesthesia***, ***involuntary spasms***/ ***cramps***. ◙ The reason for this Hypocalcemia is: 25-hydroxyvitamin D is formed in liver → Then, an enzyme called **(1-alpha-hydroxylase)** in **kidneys** converts it to the active form → **1,25-dihydroxyvitamin D**. In CKD → deficiency of 1-alpha hydroxylase → ↓ vit D and calcium. ***Example***, An elderly patient with CKD and DM presents with **loss of sensation** in his **fingers** and **muscle cramps**. His labs show: Hyperkalemia, Hypocalcemia, hyperphosphatemia, and Anemia. ◙ The likely reason for his symptoms is → **Hypocalcemia**

	◙ **IMPORTANT & TRICKY**: Sometimes the answer would be indirect, such as → **1,25-dihydroxyvitamin D3 deficiency**. "the active form of Vit. D". ◙ **Another valid answer**: Sometimes the answer would be indirect, such as → **1,25-dihydroxycholecalciferol overreplacement**. " √ In end stage kidney disease → ↓ Vitamin D → ↓ calcium → The body will try to compensate for this hypocalcemia → ↑ bone turnover rate (and ↑ parathyroid hormone to increase the reduced calcium) → with time, as PTH ↑, Ca also ↑, and this suppresses the PTH making it back to normal. It also suppresses the elevated bone turnover leading to → reduced bone turnover √ reduced bone turnover also called adynamic bone disease which causes musculoskeletal pain and immobility. In adynamic bone disease due to 1,25-dihydroxycholecalciferol overreplacement, there will be hypocalcemia + inappropriately normal PTH. **1,25-dihydroxyvitamin D3 deficiency → ↓ Calcium absorption → Hypocalcemia** Be careful, the question asks about the "**cause**" for his symptoms, which is **hypocalcemia** 2ry to **1,25-dihydroxyvitamin D3 deficiency** in this stem.
Key 18	**Autosomal Dominant Polycystic Kidney Disease (ADPKD)**

• If a patient presents with **Hematuria** (either microscopic or gross) [+] **HTN** [+] **Loin/Flank Pain.**

→ think of **Adult Polycystic Kidney Disease (ADPKD**. "**Important √**"

• Remember that **ADPKD** is associated with **Intracranial Aneurysm**! (**Important √**)

Example,

A 44 YO ♀ Known cases of Chronic Kidney Disease presents with Hypertension and loin pain. Her father died at the age of 54 due to cerebral aneurysm. What is the likely Investigation that would lead to diagnosis in this lady?

→ **Ultrasound of the Kidneys, Ureters and Bladder**.

√ You supposed to be able to know that this is a case of **ADPKD** as this disease is an **Autosomal dominant** which means that **50%** of children will be affected (Hx of father) and it is often associated with **intracranial aneurysm**.

√ Furthermore, **ADPKD** can lead to progressive **CKD**.

√ The diagnosis is made by **Ultrasound** as it can detect the cysts.

Important Table

Hematuria + HTN	Polycystic Kidney Disease (ADPKD)	Ultrasound

Hematuria + Hemoptysis	Goodpasture Syndrome	Anti-GBM Abs
Hematuria + Hemoptysis + Nasal/Sinus problems	Wegener's (Granulomatosis with Polyangiitis)	c-ANCA
Hematuria + Bloody diarrhea	Hemolytic Uremic Syndrome (HUS) (E.coli)	www.Plab1keys.com

Key 19

Hemolytic Uremic Syndrome (HUS)

Triad:

1) Hemolytic Anemia (Hemolysis)

2) Uremia (Acute Renal Failure): low urea and creatinine.

3) Thrombocytopenia (Low Platelets)

♦ ***Children***

Eating Undercooked Contaminated food → E. Coli O157 → Produce Verotoxin → Profuse Diarrhea → turns to Bloody Diarrhea → (after 2-14 days) → Uremia "Acute Renal Failure" (Hematuria, Proteinuria, ↑ Urea and Creatinine)

So, remember:

- **Diarrhea**

→ turns **Bloody**

→ **Renal Failure (namely: Acute kidney injury) √**

(Hematuria, high Creatinine…etc).

± Features of Anemia (e.g. Pallor, Fatigue).

Rx → **Supportive:**

√ **IV fluids**. √ ± **Blood Transfusion** √ ± **Dialysis** (if required)

√ If Very Severe → Plasma Exchange

◙ **Never Give Antibiotics in HUS**!

(More toxins are released as the E. Coli dies)

Note:

HUS Triad [+] **Fever** [+] **Neurological manifestations**

→ **TTP "Thrombotic Thrombocytopenic Purpura"**

Key 20

Hematuria that develops after a Hx of Upper Respiratory Tract Infection (URTI) (Sore throat/ Coryza) or less often after gastroenteritis is either due to either:

♦ **IgA Glomerulonephritis (Berger's Disease)** [**OR**]

♦ **Post-Streptococcal Glomerulonephritis.**

IgA Glomerulonephritis (Berger's Disease)	Post-Streptococcal Glomerulonephritis
1-2 days after URTI	**1-2 weeks after URTI**
Main presentation → **Hematuria**.	Main Presentation → **Proteinuria**
Usually **Young Males**	♦ Associated with ↓ **Complement levels** (C3). ♦ **Renal biopsy** → "**Humps**" on electron microscopy.
√ Organism → **Group A beta-hemolytic Streptococci** (usually **Streptococcus Pyogenes**) √ **Treatment** → **Mainly Supportive √** (the Majority resolves spontaneously).	

Key 21

Causes of Small Kidneys:

√ **Chronic Pyelonephritis.**

√ **Chronic Glomerulonephritis.**

√ **Hypertensive Renal Disease.**

√ **Bilateral Renal Artery Stenosis.**

Causes of Large Kidneys:

√ **ADPKD**. (Multiple cysts make kidneys larger)

√ **Obstructive Uropathy** (e.g. due to stone, enlarged prostate → Hydronephrosis → large kidney).

Example,

An elderly patient presents with Nausea and Prostatism (Urinary Frequency, Hesitancy, Post-void dribbling). He has HTN. U/S reveals **small** kidneys. His serum creatinine is 170 (elevated).

→ **Hypertensive renal disease**.

Hypertensive renal disease → small and scarred kidneys → Renal impairment.

Notes:

	√ **Large Prostate** e.g. Prostate cancer or Benign prostatic hyperplasia (BPH) → Features of prostatism + **Obstructive Uropathy and Hydronephrosis** (one of the causes of Large Not small kidneys. Thus, invalid answer here). √ **Reflux Nephropathy** → Dilated Pelvicalyceal system and occurs mainly in the young. (Large kidneys).
Key 22	**Again** • Simple **lower UTI** (**Dysuria**, **Frequency**) is treated by → **Trimethoprim** or Nitrofurantoin Regardless of Hx of Hypertension! • Uncomplicated **Upper UTI** (**Acute Pyelonephritis**) → **Ciprofloxacin** or **Co-amoxiclav** or **Cefalexin** (if pregnant + outpatient)
Key 23	**Scenario** A 47 YO ♂ with end-stage chronic kidney disease has had a successful renal transplant. Three days later while still in the renal ward, he develops rapid and shallow breathing, generalised oedema and he is unable to void "anuria". His Vitals and labs are as follows: HR: 118 bpm ▌BP: 182/90 ▌RR: 24 Sodium 136 (N: 135-145) ▌Potassium 6.6 (N: 3.5-5) ▌Creatinine 700 (N: 70-150)

The **BEST** management is → **Haemodialysis**.

• This is a likely case of **Transplant Rejection** or **Host-Versus-Graft**.

• Careful! Many may choose ***Calcium gluconate*** or Calcium chloride "to protect the heart" followed by ***Insulin and Glucose*** "to decrease the serum potassium". This is **NOT** the "**BEST**" management here "the question asks about the best treatment!".

• The patient has **Severe hyperkalemia** + **Anuria** + **Oedema** + **Hyperventilation**.

• The patient has also **Acidosis** (indicated by the rapid shallow breathing).

• **Lowering the potassium alone would not treat all these features**!

• Moreover, the potassium will return to the serum causing hyperkalemia again. "The patient has no functioning kidney and toxins are accumulating in his body".

• Additionally, the patient is already **overloaded** "generalised edema" with **Anuria** "cannot pass urine"; thus, **we cannot give him IV fluid**!

• Haemodialysis is supportive while we wait for another suitable kidney transplant.

• $K^+ \geq 6.5$ → Severe Hyperkalemia.

• Final tip → Calcium Chloride is mainly "but not only" given if there are ECG changes 2ry to hyperkalemia such as **Tall Tented "T" wave**, **Wide QRS complex**.

	Some Indications of Hemodialysis: √ Persistently high potassium ≥ 6.5 [Refractory Hyperkalemia]. √ Severe Metabolic Acidosis. √ Fluid overloaded with Anuria or non-effective diuretics. √ Uremic Pericarditis, Pulmonary edema. √ Uremic Encephalopathy
Key 24	***Scenario*** 52 YO man presents to the A&E with fever, rigors and vomiting for 2 days. There is severe tenderness over the right costovertebral angle. His Urinalysis is positive for blood, nitrites and leucocyte esterase. Creatinine: Normal ▌Urea: 10 (high) ▌WBCs: High ▌CRP: High ▌K^+: Normal. He was immediately commenced on IV antibiotics and IV fluid. The most appropriate Investigation → **Urine Culture and sensitivity.** **NOT U/S.** • **This is likely a case of Acute Pyelonephritis.** • Remember: (**loin/ back pain**, **fever**, **rigors**), [±] Urinalysis shows blood, nitrites, leucocyte esterase → **Acute Pyelonephritis**.

	• Here, the question mentions "**Costovertebral angle tenderness**" instead of "**Loin, back or Flank pain**". They are nearly the same as the costovertebral angle is at the (back, towards the flank). • The investigations for Acute Pyelonephritis are: Urinalysis → then, Urine Culture. • It is preferred to take the urine sample for culture before starting the IV antibiotics. Nonetheless, they have started the antibiotic before the culture in this stem. • Remember, in suspicious meningitis, we start empirical IV antibiotics even before LP or blood culture.
Key 25	**Middle aged man with 2 episodes of microalbuminuria. BP 128/70 mmHg. Normal renal functions. He is overweight. What additional therapy would benefit this patient?** a. **ACE-I** b. Statins c. B-blockers d. diuretics ACE inhibitors are reno-protective (i.e. they delay the progress of CKD).

Key 26	**A 5-year-old girl with 5 days history of bloody diarrhoea and dehydration. Blood culture shows E. coli** **Na+ low, K+ high, Creatinine high, Urea high, Calcium Normal, Bicarbonate low. What is the like diagnosis?** a. **Acute kidney injury** b. Addison's c. Renal tubular acidosis √ This is a classic case of HUS (Hemolytic-Uremic Syndrome). √ **HUS** is the **most common** cause of **acute kidney injury** in **children** and is increasingly recognized in adults
Key 27	**A 4-year-old boy with recurrent UTI. What is the most likely anatomical abnormality?** A. **Vesicoureteral reflux** B. Urethral valve C. Horse shoe kidney D. Bladder cancer --- **5-year-old Girl with recurrent UTI (has had 3 UTIs) responsive to antibiotics.**

Next Investigation of choice?

a. MCUG

b. **Renal USS**

c. DMSA

d. Urine mcs

e. CTSCAN

Reflux Nephropathy

• Urine goes back from bladder to ureters and kidneys (Vesico-Ureteric Reflux) → Dilated Pelvicalyceal system → **Repeated UTIs** → Progressive **Renal Failure**.

• Occurs mainly in the young (**children**).

♦ An important cause → Congenital abnormality of the insertion of ureters into the urinary bladder (can be seen on US).

♦ **Dx**

√ Initial → Renal Ultrasound (+) Urinalysis, urine culture and sensitivity.

√ Gold standard → Micturating Cystourethrogram.

	√ For parenchymal damage (cortical scars) → Technetium Scan (DMSA). ♦ **Rx** √ Initially → Low-dose antibiotics prophylaxis (trimethoprim) daily. √ Failed? Or Parenchymal damage? → Surgery (Ureters Re-implantation).
Key 28	**A 5 YO child presents with ↑ weight, Puffy eyes, Lower limb swelling. He is otherwise healthy and happy. What is the DEFINITIVE diagnostic modality?** → **Renal Biopsy** **In Nephrotic Syndrome:** **√ Renal biopsy (Definitive Test) → electron microscopy shows fusion of podocytes.**
Key 29	**A 69yr old present to emergency department with thirst, anorexia & lethargy. He is a known hypertensive not on regular medication or follow up. OE, blood pressure was 170/100, Urea-high, Creatinine- high, USG shows bilateral small kidneys. What is the most likely cause?** A. **Hypertensive nephropathy** B. BPH

C. Prostate car

D. Chronic Pyelonephritis

E. VUR

Causes of Small Kidneys:

√ **Chronic Pyelonephritis.**

√ **Chronic Glomerulonephritis.**

√ **Hypertensive Renal Disease.**

√ **Bilateral Renal Artery Stenosis.**

Causes of Large Kidneys:

√ **ADPKD**. (Multiple cysts make kidneys larger)

√ **Obstructive Uropathy** (e.g. due to stone, enlarged prostate → Hydronephrosis → large kidney).

Example,

An elderly patient presents with Nausea and Prostatism (Urinary Frequency, Hesitancy, Post-void dribbling). He has HTN. U/S reveals small kidneys. His serum creatinine is 170 (elevated).

→ **Hypertensive renal disease**.

	Hypertensive renal disease → small and scarred kidneys → Renal impairment. **Important**, When you see **Hypertension** + Hx of **repeated urinary tract infections** (**UTIs**) → Think of: **CHRONIC Pyelonephritis** **Repeated UTIs → Renal Scarring → Chronic Pyelonephritis → Hypertension**
Key 30	**15yr old with facial & ankle swelling. Urinalysis shows protein +++. What is the likely cause?** A. **Minimal change GN** B. Membranous GN C. Post streptococcal GN D. Ig A nephropathy ◙ **Minimal change disease** nearly always presents as **nephrotic syndrome**, accounting for 75% of cases in children and 25% in adults. ◙ Around 80% of cases in children are due to **Minimal change glomerulonephritis**.

	◙ The condition generally carries a **good prognosis** with around 90% of cases responding to **high-dose oral steroids**.
Key 31	**A patient going for CT Scan with contrast. How can you reduce contrast renal injury?** → **IV Fluids (0.9% Normal saline)**. In CT scan with **contrast**, to reduce the risk of "**Contrast Induced Nephropathy**" → **Drink Plenty of Fluids**. Another correct answer → **IV fluid normal saline (0.9% NaCl)** pre and post procedure is especially in high risk patients such as elderly with DM. • Additional Note, **metformin** is **Nephron-harmful** and thus needs to be ceased "**stopped**" before any **contrast** study.
Key 32	**An elderly patient with CKD (Chronic Kidney Disease) and DM presents with loss of sensation in his fingers and muscle cramps. His labs show:** **Hyperkalemia, Hypocalcemia, hyperphosphatemia, and Anemia.**

◙ The likely reason for his hypocalcemia is

→ **1,25-dihydroxyvitamin D3 deficiency**. "the more active form of Vit. D".

◙ Patients with late **CKD** usually have **Hypocalcemia** which results in ***tingling***, ***numbness***, ***paraesthesia***, ***involuntary spasms***/ ***cramps***.

◙ The reason for this Hypocalcemia:

25-hydroxyvitamin D is formed in liver → Then, an enzyme called **(1-alpha-hydroxylase)** in **kidneys** converts it to the active form → **1,25-dihydroxyvitamin D**.

Patients with CKD may lack this enzyme and therefore, this conversion does not occur.

1,25-dihydroxyvitamin D3 deficiency → ↓ Calcium absorption → Hypocalcemia

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√ **25**-alpha-hydroxylation of vit. D occurs in → **Liver**.

√ **1**-alpha-hydroxylation of vit. D occurs in → **Kidney**.

25-hydroxyvitamin D is formed in liver → Then, an enzyme called **(1-alpha-hydroxylase)** in **kidneys** converts it to the active form

→ 1,25-dihydroxyvitamin D.

	So, why is there vitamin D deficiency in chronic renal failure? → **due to reduced activity of 1-alpha-hydroxylation**. **Mnemonic** (**K**) before (**L**) in alphabetics. (**1**) before (**25**) **K**idney → **1** ▌**L**iver → (**25**) **Another mnemonic** **1K** **1=Kid**
Key 33	**7-year-old boy with increased weight and bilateral swollen legs. No other complains. Next diagnostic investigation?** A. TFT B. X-ray C. Blood glucose D. **Urinalysis** E. LFT

	He likely has Nephrotic Syndrome. We need to see protein in urine. So, urinalysis is the right answer. Remember, Nephrotic Syndrome = **Proteinuria** + **Albuminemia** + **Oedema**. Thus, **24-hour protein in urine (Urinalysis)** + **serum albumin** levels are required to help in diagnosis. The most **definitive** Dx → Renal Biopsy.
Key 34	**A 23-year-old man was found lying on the floor behind his flat. He is a known intravenous drug user and has not been seen for 3 days. Results of his investigation are as follows: Urea – 45mmol/L, Creatinine 1200 micromole/L What in–estigation would confirm the cause of his renal impairment?** A. Abdominal Ultrasound scan B. Blood cultures C. **Serum creatinine phosphokinase** D. Echocardiograph E. TFT **Rhabdomyolysis**

• As skeletal muscles are dying → they release (**Myoglobulin**, **Potassium**, **Creatine Kinase**…).

• **Common Scenarios and Hints: (Important √)**

√ A person was trapped for several hours under a heavy object.

√ A fall followed by a long period of lying on the floor.

√ An elderly with frequent falls presents with Acute kidney injury.

√ IV drug abuser was found on the floor not moving for a long period.

√ Long-distance run (e.g. Marathon runner) "Severe Exertion/ Severe Dehydration".

√ Severe Crush injury

√ Exercise-induced rhabdomyolysis (e.g. in athletes)

± Hematuria (**Reddish Brown** or **Tea-coloured urine**) (False Positive as the cause of redness is myoglobulin (which has heme), while RBCs are not found in urine dipstick)

± Hypotension

± AKI (High urea and Creatinine)

± Very high CK (Creatine Kinase)

• **Myoglobulin** is **nephrotoxic** and thus can lead to **Acute Kidney Injury (AKI).** Therefore, rehydration with **IV fluid** is an essential initial step. That's why Rhabdomyolysis is a medical emergency that you have to be aware of!

	• **ECG** must be performed as the released potassium from the dying muscles (**hyperkalemia**) can be dangerous. If ECG changes suggesting hyperkalemia (**Tall tented T wave, Wide QRS**) are found: → Protect the heart by giving **IV Calcium Chloride** or IV **Calcium Gluconate** **before anything else!** ◙ **Important points on Rhabdomyolysis:** √ Main Complications of Rhabdomyolysis → **AKI** and **Hyperkalemia**. √ Initial management → **IV fluid** (to try to avoid acute kidney injury). √ Initial Investigation for management → **ECG** √ If **Tall T wave, Wide QRS**, the initial line → give **IV calcium chloride**/ **gluconate**. √ The best initial test that is **specific for Rhabdomyolysis** → **Urine analysis** → Reddish-brown (Tea-coloured) → Falsely +ve hematuria. √ **To confirm** → √ **CPK level** (**Creatine Phosphokinase**) "it indicates muscle necrosis". √ Other lines of treatment include: Sodium Bicarbonate ▌Dialysis (in severe cases)
Key 35	**A hypertensive patient was commenced on a medication. After that, he developed proteinuria (+3) and renal function impairment. What is the causative medication?** → **ACE inhibitor**.

	√ Deterioration of Renal function tests after initiation of **ACE inhibitor** in a hypertensive patient means that the patient already has → **Bilateral Renal Artery Stenosis** which was responsible for his hypertension. So, ACEIs are contraindicated in bilateral renal artery stenosis. √ **Bilateral Small Kidneys + Hypertension** → **Bilateral renal artery stenosis** → **Or: hypertensive renal disease**
Key 36	**A female presents with dysuria, loin pain and rigors.** Likely → **acute pyelonephritis**. Initial investigation → **urinalysis**. Then → **urine culture**.
Key 37	***Hematuria that develops after a Hx of Upper Respiratory Tract Infection (URTI) (Sore throat/ Coryza) or less often after gastroenteritis is either due to:***

♦ **IgA Glomerulonephritis (Berger's Disease)** [OR]

♦ **Post-Streptococcal Glomerulonephritis.**

IgA Glomerulonephritis (Berger's Disease)	*Post-Streptococcal Glomerulonephritis*
1-2 days after URTI	**1-2 weeks after URTI**
Main presentation → **Hematuria**.	Main Presentation → **Proteinuria**
Usually **Young Males**	♦ Associated with ↓ **Complement levels** (C3). ♦ **Renal biopsy** → "**Humps**" on electron microscopy.

Key 38

◙ 2 YO child was noticed by his mother to be gaining weight regardless of his poor feeding. There is gradual swelling of his face, feet and legs. The child feels fatigue and his urine is noted to be *foamy*.

The likely Dx → **Nephrotic Syndrome**.

√ The most appropriate Investigation → **24-Hours urinary protein**.

	√ The most **definitive** diagnostic test → **Renal Biopsy** Remember, Nephrotic Syndrome = **Proteinuria** + **Albuminemia** + **Oedema**. Thus, **24-hour protein in urine** + **serum albumin** levels are required to help in diagnosis. • Note, one important explanation of **foamy urine** is the presence of **high protein** in it.
Key 39	◙ 24 YO man was rescued after crush injury in a car accident. He has dark urine with impaired creatinine and urea. His BP is 100 mmHg and HR is 125 bpm. • The likely cause of his renal failure → **Myoglobin** **A case of Rhabdomyolysis** Myoglobin is reno-toxic.
Key 40	**A patient with history of chronic glomerulonephritis and diabetes presents for check-up. He is on metformin. His BP is 144/91.** **His creatinine is 151 (normal: 70-150).** **Urea is 7.2 (normal: 2-7).**

	eGFR is 37 (normal >90). **What medication can delay the progression of his renal impairment?** → **ACE inhibitor (e.g., ramipril, enalapril).** "Reno-protective. However, avoid using it if eGFR is < 30".
Key 41	There are 4 medications that need to be stopped if a patient presents with **Diarrhea**/ Vomiting (Risk of **Dehydration** and **AKI**) until symptoms resolve: **DAMN Drugs** 1) **Diuretics** (e.g. Furosemide, bendroflumethiazide) → (↑ dehydration). 2) **ACEE inhibitors** (e.g. enalapril, ramipril) and **ARBS** (e.g. Losartan) → (AKI). 3) **Metformin** → (↑ lactic acidosis in a patient with dehydration). 4) **NSAIDs** → (AKI) Risk of **D**ehydration (**D**iarrhea/vomiting) → stop **D**AMN. --- **◙ Important causes for AKI "Acute Kidney Injury" to remember:** √ **Rhabdomyolysis**. (prolonged immobilisation, trapped under a heavy rock…).

	√ **HUS** (usually a child with bloody diarrhea then hematuria and KFTs impairment). √ **Dehydration**: diarrhea, vomiting (causes of hypovolemia). √ **Sepsis**. √ Following **major surgery**. √ **DAMN** Drugs: **D**iuretics, **A**CEi and **A**RBS, **M**etformin, **N**SAIDs.
Key 42	An elderly man is brough by his son to the GP surgery as he complains of severe lethargy, nausea, loss of appetite and weight loss. His medical Hx includes HTN. His BP is 170/115. His renal function tests found to be abnormal with high urea and creatinine and GFR of 40. US of kidneys is performed and reveals reduced cortical thickness and reduced renal parenchymal volume of both kidneys. What is the most likely Dx? → **Hypertensive nephropathy**. **Hypertensive Nephropathy** • Renal failure 2ry to HTN. If left untreated → ENKD can develop. • **Features** → Fatigue, Nausea, Anorexia, Weight Loss, HTN.

	• **US** → Small kidneys (reduced parenchymal volume and cortical thickness bilaterally). √ It is important to do **US** to exclude bilateral renal artery stenosis.
Key 43	**All patients with Diabetes + Microalbuminuria should be started on ACE inhibitor (Angiotensin- Converting Enzyme Inhibitors) such as enalapril, lisinopril…etc EVEN IF NORMOTENSIVE.** √ DM can lead to diabetic nephropathy. √ ACEi is reno-protective and would delay the kidney damage.

Key 44	**A 69yr old present to emergency department with nausea, loss of appetite & lethargy. He is a known hypertensive not on regular medication or follow up. OE, blood pressure was 170/100, Urea-high, Creatinine- high. He is anaemic and cachectic. His Ca++ is low, creatinine is high, and PSA is 4.5. What is the most likely cause?** A. **Hypertensive nephropathy** B. BPH C. Prostate car D. Chronic Pyelonephritis E. VUR

	• Lethargy, loss of appetite and nausea are seen in hypertensive nephropathy. • If left untreated, hypertensive nephropathy can lead to end-stage kidney disease (ESKD). • Prostate cancer is a distractor here as prostate cancer can cause hypercalcemia (here it is hypokalemia due to ESKD) and in prostate cancer, PSA would be very high when the patient reaches the level to be cachectic.
Key 45	**60 YO man with stage 4 chronic kidney disease on antihypertensive medications presents with the following:** **BP: 160/100** **Urinalysis: Protein 1+, Blood 1+** **Urea: 8** **Creatinine: 240** **eGFR: 25** **U/S: reduced renal parenchymal volume, reduced cortical thickness bilaterally.** **What is the most likely diagnosis among the options?** **A) Chronic pyelonephritis.** **B) Unilateral renal artery stenosis.** **C) Glomerulonephritis.** **D) Rhabdomyolysis.** **E) Acute kidney injury.**

	• The stem mentions "bilateral small kidneys". • Options A, B, and C can cause small kidneys. In addition, hypertensive nephropathy can also cause bilateral small kidneys but not mentioned here. • Option A (chronic pyelonephritis) is not suitable here as there is no mention of Hx of recurrent infections and scaring. • Option B (unilateral renal artery stenosis) can cause "unilateral" small kidney. Thus, it is wrong. • The most suitable answer here is option **C → Glomerulonephritis**.
Key 46	**A 23 YO man found to have microscopic haematuria on a routine check-up. His father has chronic kidney disease and a previous haemorrhagic stroke. His grandfather on his paternal side dies from intracranial haemorrhage. His BP is 155/105.** **The investigation that most likely would lead to a diagnosis is:** → **Ultrasound of kidneys, ureters and bladder (US of KUB)**. √ He is likely having autosomal dominant polycystic kidney disease (ADPKD). √ Hematuria + HTN + Strong FHx of hemorrhagic stroke (remember that intracranial aneurysm is an important association with ADPKD). **√ IMPORTANT:**

	ADPKD is diagnosed by (U/S of KUB) not by (Genetic testing) as genetic testing for ADPKD is particularly difficult to interpret and we resort to it only if U/S is negative and the suspicious of ADPKD is high.
Key 47	**A 75 YO woman presents with severe low back pain and pelvic pain. She has end-stage kidney disease, type 2 DM, HTN, and ischemic heart disease and she is on medications for these diseases. X-ray has been done and showed no fractures. Blood tests revealed low serum calcium, high phosphate, and normal parathyroid hormone. What is the most likely cause for her presentation?** → **1,25 dihydroxycholecalciferol overreplacement.** √ In end stage kidney disease → ↓ Vitamin D → ↓ calcium → The body will try to compensate for this hypocalcemia → ↑ bone turnover rate (and ↑ parathyroid hormone to increase the reduced calcium) → with time, as PTH ↑, Ca++ also ↑, and this suppresses the PTH taking it back to normal. It also suppresses the elevated bone turnover leading to → reduced bone turnover √ reduced bone turnover also called adynamic bone disease which causes musculoskeletal pain and immobility. In adynamic bone disease due to 1,25-dihydroxycholecalciferol overreplacement, there will be hypocalcemia + inappropriately normal PTH. √ 1,25-dihydroxycholecalciferol is the active form of vitamin D.

Key 48	**Important**: "somehow related to the last key" **What if the patient with end-stage kidney disease presents with musculoskeletal pain and hypocalcemia and low Vitamin D and still high PTH?** This patient is having **2ry hyperparathyroidism** (still high PTH to try to increase the low calcium, i.e., still high bone turnover). This means he is having high bone turnover and even though PTH is high to try to elevate the serum calcium caused by the chronic kidney disease, it still is not enough to compensate the vitamin D. The diagnosis here is **2ry hyperparathyroidism** or **vitamin D deficiency**. Both are correct. This patient would benefit from → **Vitamin D supplements**.
Key 49	Initial Rx for (**Hypocalcemia**) eg, **tingling sensation, low serum calcium:** → Calcium gluconate infusion. (*Remember that end-stage renal failure can cause hypocalcemia. If severe and left untreated it can cause arrhythmia, cardiac arrest and death*).

Key 50

Q1) A 27 YO woman presents with 1-week complaints of strong desire to empty her bladder, lower abdominal discomfort, stinging during urination and an increased number of urinations. She is not sexually active. She is afebrile. There is no loin pain. Her urinalysis shows leucocytes and nitrates.

- The most likely Dx → **Lower UTI**.
- The most appropriate Rx → **Nitrofurantoin**.

Q2) A 27 YO woman presents with 1-week complaints of strong desire to empty her bladder, lower abdominal discomfort, stinging during urination, right loin tenderness, fever, rigors. Urinalysis shows leucocytes and nitrates.

- The most likely Dx → **Acute Pyelonephritis**.
- The most appropriate Rx → **Co-amoxiclav or Ciprofloxacin**.

Summary:

√ **Dysuria**, **frequency**, **urgency ± lower abdominal Pain** → Lower UTI

→ *Trimethoprim* (**or**) *Nitrofurantoin*. **If pregnant → Cefalexin.**

√ **The above** + **Loin** or **Back** pain + **fever** → Acute pyelonephritis (Upper UTI)

→ *Ciprofloxacin* (**or**) *Co-amoxiclav*.

Key 51

A 60 YO woman presents complaining of blood in urine that she has noticed today. She had sore throat last week and was given antibiotics. She has also developed maculopapular rash on her trunk. Her body temperature is 37.9. Urea and creatinine are high.

The most likely Dx → Acute interstitial nephritis.

◙ **Allergy** (e.g., **drug intake** followed by **rash**, **fever**) + **Hematuria** + **↑ creatinine**.

Think → **Acute interstitial nephritis**.

◙ Massive **hemorrhage** (e.g., during surgery) and **hypotensive shock** + High creatinine

Think → **Acute Tubular Necrosis** (**ATN**)

(ATN is the commonest renal cause of acute kidney injury).

√ "The kidneys need to remain well hydrated/ perfused to avoid acute "**tubular**" necrosis".

√ "Prolonged **ischemia → "low perfusion"** to kidneys → **Dying tubules** = **necrosis** = **Acute tubular necrosis** → AKI"

Key 52	**Important Summary Points – Nephrology:** ◙ Antibiotic intake (eg, for URTI) followed by maculopapular rash + HEMATURIA + fever + Deranged kidney function tests (KFTs) → **Acute interstitial nephritis**. ◙ Massive hemorrhage (eg, during surgery), hypotensive shock, Deranged KFTs → **Acute tubular necrosis**. ◙ URTI or GE, 1-2 days later presents with hematuria especially in young patients + Deranged KFTs. → **IgA glomerulonephritis (Berger's disease)**. ◙ URTI or GE, 1-2 weeks later presents with Proteinuria "protein in urine" ± Hematuria + Deranged KFTs. → **Post-streptococcal glomerulonephritis**.
Key 53	**A 70 YO man presents to the A&E complaining of difficulty breathing especially when lying flat. He has end-stage renal failure and is on hemodialysis. However, he has missed the last few hemodialysis appointments. His creatine and urea are markedly elevated. His serum potassium is 7 (normal: 3.5-5 nmol/L). His daily urine output is less than 40**

	ml (ie, he has anuria). His chest X-ray shows pulmonary oedema. What is the most appropriate management? → **Urgent Haemodialysis**. √ Don't give furosemide to patients with severe renal impairment + Anuria! √ This patient with uremia and severe hyperkalemia and fluid overload needs urgent hemodialysis.
Key 54	**A 48 YO man presents to a renal clinic complaining of frothy urine (ie, proteinuria) + lower limm oedema. He is diabetic and hypertensive. His blood pressure is within normal ranges. His labs:** **Urine Protein:Creatinine ratio is 490 (normally < 350 mg/mmol).** **HbA1C is 45 (normally < 48 mmol/mol).** **What is the most likely cause for his proteinuria?** → **Membranous Nephropathy**. • It is **not** diabetic nephropathy (his HbA1C is normal. Ie, his DM is controlled). • It is **not** hypertensive nephropathy (His BP is normal ie, controlled). • Low urine protein to creatinine ratio + proteinuria + LL edema → think **Nephrotic syndrome**.

- And the commonest cause for nephrotic syndrome in adults is **Membranous nephropathy**.

◙ The most common cause of **Nephrotic Syndrome** in **Adults**:

√ in Caucasian ▌White people ▌or if unspecified ethnicity → **Membranous GN**.

√ in Africans ▌Black ▌Americans ▌Hispanics → **Focal Segmental GN**.

◙ Generally, for PLAB 1, the most common cause of **Nephrotic Syndrome** in **Adults** (especially > 40 YO) → **Membranous GN**.

◙ And in children (80% of nephrotic S) → **Minimal change glomerulonephritis**.

Membranous glomerulonephritis

- Presentation: proteinuria / nephrotic syndrome / chronic kidney disease
- Cause: idiopathic (Mainly), infections, rheumatoid drugs, malignancy
- **Prognosis**:

♦ 1/3 of the cases → Remission.

♦ 1/3 of the cases → Partial Remission.

♦ 1/3 of the patients → progress into End-stage Renal Failure.

Key 55	One of the important complications of **CKD** is **Anemia** (eg, extreme fatigue, sleepiness, pallor). "Due to impaired iron absorption and erythropoietin production" ◙ In patients with **CKD** and **Anemia** but **with normal serum Ferritin and iron** Give → **Erythrocyte stimulating agents**. *They don't need iron*. ◙ In patients with **CKD** and **Anemia** + **Low serum Ferritin/ iron** Give → **Iron supplements followed by erythrocyte stimulating agents**.
Key 56	**A 60-year-old man with diabetes mellitus type 2 and hypertension on metformin and losartan presents complaining of lower limb edema. His urine is frothy. Fundoscopy is unremarkable (no retinopathy). His lab results are:** **Hemoglobin 121 g/L. (130-180).** **Sodium 133 mmol/L (135-146).** **Potassium 5.0 mmol/L. (3.5-5.3).** **Urea 22 mmol/L (2.5-7.8).** **Creatinine 135 mmol/L (45-95)** **HbA1c 50 mmol/mol (<48).** **Protein:Creatinine ratio 300 mg/mmol (<50).** **What is the most likely diagnosis?** **A) Diabetic nephropathy. Or:**

	B) Membranous nephropathy. • The patient has features of nephrotic syndrome: Edema, proteinuria (Protein:Creatinine ratio of 300 equals = 3-gram protein in urine in 24 hours). • The commonest cause for nephrotic syndrome in adults is membranous nephropathy. • Also, HbA1c of 50 mmol/mol is not a big number to cause diabetic nephropathy. This means, his diabetes is controlled! • Also, there is no retinopathy here, and usually, nephropathy occurs after retinopathy (however not always, but it a helping hint). • Also, idiopathic membranous nephropathy is common in diabetic patients. ◙ Therefore, the likely Dx here is → Membranous nephropathy.
Key 57	**A 60 YO man with hypertension and overactive bladder is on amlodipine (for HTN) and oxybutynin (for overactive bladder) and his BP is still high (150/95). His creatinine and urea are elevated. His GFR is 38 and his ACR is 40. What should be done?** → **Add ACE inhibitor (eg, ramipril) or ARBs (eg, losartan).** • We **add** ACEi to the already being taken amlodipine. • This is **step 2** in managing hypertension (**using 2 drugs if 1 drug is not controlling the hypertension**).

	• Since he is > 55 YO, he was started on amlodipine; a calcium channel blocker. However, his HTN is not controlled. Thus, we go to step 2 to by **ADDING** another anti-hypertensive, which is ACEi or ARBs. • Being with **chronic kidney disease** and **proteinuria** (**ACR > 30**) also supports the addition of **ACEi** or **ARBs** as they can slow the deterioration of kidney functions. (**ACEi** (eg, ramipril, lisinopril) or **ARBs** (eg, losartan) are the preferred drug for Hypertensive patients of any age/ ethnicity with **chronic kidney disease** as long as **GFR > 30** and **urine Albumin: Creatinine Ratio (ACR) > 30** [**They slow the progression of CKD**]. **√** A ratio of albumin (mcg/L) to creatinine (mg/L) [**ACR**] of **< 30** is **normal**. **√** A ratio of 30-300 signifies **microalbuminuria** (**proteinuria**) and values above 300 are considered as **macroalbuminuria**. (Albumin is a protein). Hypertension with CKD + **proteinuria** is best treated by adding **ACEi** or **ARBs**. Also, in **DM** with HTN → ACEi or ARBs.
Key 58	**When should a GP refer a patient to a Nephrologist?** • **If eGFR becomes < 30.** • **If eGFR declined by ≥ 25% within 12 months + a change of CKD category.**

	• **If eGFR declined by ≥ 15 units within 12 months (sustained ↓).** • **If ACR ≥70 mg/mmol (unless diabetic).** **Example:** **A 48-year-old diabetic patient presents for review in the GP surgery. Lab test have been made and compared to his lab results of one year ago. The eGFR was 48 a year ago, now it is 22. The other renal function tests are normal. What is the most appropriate next step?** → **Refer to nephrology.** Here, eGFR has become <30 And also, there is a decline of more than 15 units in 12 months.
Key 59	**If you suspect Nephrotic Syndrome (a child with facial swelling followed by total body swelling; ↑ in weight, scrotal edema, LL pitting edema)** **The initial step is → urinalysis** **(Urinalysis** initially to check for **proteinuria** and then do more investigation eg, **serum albumin level**. **Renal biopsy** is diagnostic and in **children** the most common type is **minimal change** nephrotic syndrome**).**

	◙ **In nephrotic syndrome, remember the triad of:** 1. **Proteinuria** (> 3g/24hr). 2. **Hypoalbuminaemia** (< 30g/L) and 3. **Oedema** **Other possible findings** → frothy/ foamy urine, fatigue, ↑ weight, loss of appetite. **Important:** **The anatomical structures affected in nephrotic syndrome are** → **Glomeruli.** **Glomeruli** are small structures – small blood vessels that filter the blood from unwanted substances and keep important substances such as proteins in it. When damaged as in nephrotic syndrome, they throw proteins (mainly albumin) in the urine, causing proteinuria and hypoalbuminemia and as a result, oedema.
Key 60	◙ URTI (sore throat, enlarged tonsils). or GE (Gastroenteritis) 1-2 days later presents with hematuria especially in young patients ± Deranged Kidney function tests (with or without).

	→ **IgA glomerulonephritis (Berger's disease)**. Management (imp): • If normal blood pressure, low proteinuria (<50 urine protein: creatinine ratio): → Supportive Rx (continue to observe). **√** • If high blood pressure OR Significant proteinuria (>50 mg/mmol uPCR) → ACE inhibitor. • If high proteinuria (> 1g/ day) or (<50 mg/mmol uPCR) → Prednisolone can also be used.
Key 61	**NICE guidelines suggest:** Patient with chronic kidney disease should take lipid modification agents (eg, atorvastatin). This is despite their lipid profile results and without the need of a formal risk assessment.
Key 62	**Mineral Bone Disease**

◙ Mineral Bone disease is seen in late-stages chronic kidney disease (stage 4 and above).

◙ **Basic problems in chronic kidney disease (CKD):**

√ 1-alpha hydroxylation normally occurs in the kidneys

→ CKD leads to **low vitamin D**.

√ The kidneys normally excrete phosphate → CKD leads to **high phosphate**.

◙ **This, in turn, causes other problems:**

√ The high phosphate level 'drags' calcium from the bones, resulting in osteomalacia.

√ Low calcium: due to lack of vitamin D, high phosphate

√ Secondary hyperparathyroidism (**↑ PTH**): due to low calcium, high phosphate and low vitamin D.

◙ Management:

In the exam, to address -treat- the biomedical abnormalities, pick:

→ Calcium and Vitamin D supplements (eg, AdCal-D3).

The aim of Rx is to reduce phosphate and parathyroid hormone levels.

- Vitamin D or its analogue eg, Alfacalcidol, calcitriol + Calcium supplements.
- Reduced dietary intake of phosphate is the first-line management.

• Phosphate binders: eg, sevelamer (a non-calcium-based binder that is now increasingly used).

• Parathyroidectomy may be needed in some cases.

Scenario:

A patient with CKD that has become a late stage (4 or more) presents with: ↓calcium, ↑phosphate, ↑PTH (secondary not tertiary hyperparathyroidism)

• Think → Mineral Bone Disease.

• What is the type of hyperparathyroidism here (↑PTH)?

→ Secondary hyperparathyroidism (low or N. calcium, high or N. phosphate).

• What is the most likely cause of these blood abnormalities?

→ Vitamin D deficiency.

• Why not tertiary hyperparathyroidism (as it can be cause by end-stage CKD)?

→ In tertiary hyperparathyroidism, calcium would be high (↑) not low as here.

• What is the most appropriate management to address these abnormalities?

→ Calcium and Vitamin D supplements (eg, AdCal-D3).

Remember the differences between hyperparathyroidism types:

hyperparathyroidism			
	Primary hyperparathyroidism	**Secondary hyperparathyroidism**	**Tertiary hyperparathyroidism**
PTH	↑	↑	↑↑↑
Ca2+	↑ *	↓/N	↑
P04+	↓	↑/N	↑
Vitamin D	N/↓	↓↓↓	↓/N
Comments	In 85% a solitary parathyroid adenoma is present Important differential diagnosis is FHH (see Table 3)	Causes include: • chronic renal failure • vitamin D deficiency	Caused by: • end stage renal failure

* Differential diagnosis of hypercalcaemia includes drug induced (eg. lithium, thiazides) and hypercalcaemia of malignancy

Key 63

Important notes on SGLT2 Inhibitors (Flozin Family):

• In addition to ACE inhibitors → **Sodium-Glucose Co-Transporter-2** (**SGLT2**) **inhibitors** provide **renal**, **cardio**, and **diabetic** protection.

• Thus, you can add it to ACE inhibitor if needed (eg, if HbA1c is still > 48 mmol/mol).

• SGLT2 inhibitors are beneficial in patients with Type 2 diabetes + **cardiovascular disease**/ **risk**.

• Examples of **SGLT2 inhibitors** → **flozin** → Dapagliflozin, Canagliflozin, Empagliflozin.

Key 64

Scenario

A 55-year-old woman presents to the clinic for a routine follow-up visit. She has type 2 diabetes mellitus. Her recent HbA1c is 44 mmol/mol (<42). Eye examination show no evidence of retinopathy. Her blood pressure is 140/90 mmHg. Her BMI is 32 kg/m^2. Her urine is frothy. Urinalysis shows significant proteinuria. Her urine albumin-to-creatinine ratio (ACR) is markedly elevated at 46 mg/mmol. What is the most likely cause of the frothy urine in this patient?

A) Diabetic nephropathy.

B) Hypertensive nephropathy.

C) Membranous nephropathy.

D) Polycystic kidney disease.

E) Acute interstitial nephritis.

• It is not **diabetic nephropathy** because:

√ The HbA1c is relatively well-controlled.

√ Also, she does not have diabetic retinopathy, which usually occurs before diabetic nephropathy.

• It is mot **hypertensive nephropathy**. This is because the blood pressure is not elevated to a degree that causes nephropathy.

• No points in the stem support a diagnosis of Polycystic kidney disease or acute interstitial nephritis.

• The correct answer here is (**B**) → Membranous nephropathy.

Membranous nephropathy:

• (Thickening of the glomerular basement membrane)

• It is the most common cause of idiopathic Nephrotic Syndrome in Adults (especially > 40 YO).

• Presentation: proteinuria / nephrotic syndrome / chronic kidney disease

• Cause: idiopathic (Mainly), infections, rheumatoid drugs, malignancy

• Prognosis:

♦ 1/3 of the cases → Remission.

♦ 1/3 of the cases → Partial Remission.

♦ 1/3 of the patients → progress into End-stage Renal Failure.

What is the most definitive "diagnostic" investigation?

→ Renal biopsy.

Key 65

Haemodialysis in Emergency Settings

Introduction: Hemodialysis is a critical intervention in emergency settings to correct life-threatening complications arising from renal failure. It involves the removal of waste products and excess substances from the blood when the kidneys can no longer perform these functions adequately.

Indications for Emergency Hemodialysis:

1. **Hyperkalemia**:
   - Particularly urgent when serum potassium levels exceed 6.5 mmol/L or even lower levels if accompanied by ECG changes such as tall, peaked T-waves.
   - Immediate treatment may include intravenous calcium to stabilize the cardiac membrane and insulin plus dextrose to drive potassium back into cells.
2. **Acute Pulmonary Edema**:
   - Occurs due to fluid overload that is unresponsive to conventional medical treatments such as diuretics.
3. **Metabolic Acidosis**:
   - Severe acidosis (pH < 7.1) that does not respond to standard medical management, requiring immediate correction.
4. **Uremic Complications**:
   - Including conditions such as pericarditis, which result from the accumulation of uremic toxins in the body.

Procedure and Preparation:

- **Vascular Access**:
  - Temporary access via a central venous catheter is typically required to initiate hemodialysis swiftly in an emergency setting.
- **Time and Coordination**:
  - The process involves coordination with a renal team, preparation of the dialysis machine, and ensuring adequate vascular access, which can take from 30 minutes to several hours depending on the patient's condition and available resources.

Scenario Example:

A 65-year-old woman presents to the Emergency Department with severe fatigue and shortness of breath. She has a history of hypertension and chronic kidney disease. On examination, her heart rate is irregular, and she is noticeably confused. An ECG shows tall, peaked T-waves. Blood tests reveal the following results:

- Sodium: 132 mmol/L (135-145)
- Potassium: 7.1 mmol/L (3.5-5)
- Urea: 25 mmol/L (2.0-7)
- Creatinine: 480 µmol/L (70-150)
- eGFR: 18 mL/min (>90)

The decision is made to commence emergency hemodialysis. Which of the following is the most appropriate reason for initiating hemodialysis in this patient?

A) High serum creatinine.

B) Hyperkalemia.

C) Low eGFR.

D) Hyponatremia.

E) Toxin removal.

Answer → B) **Hyperkalemia** is the primary indication for initiating emergency hemodialysis in this scenario due to the immediate risk posed by high potassium levels and the associated ECG changes.

Immediate treatment may include **intravenous calcium** to stabilize the cardiac membrane and **insulin plus dextrose** to drive potassium back into cells.

Why Not the Other Options?

1. **High Serum Creatinine**:
   - Elevated creatinine indicates poor kidney function but is not an emergency indication for hemodialysis unless accompanied by severe symptoms or complications like hyperkalemia.
2. **Low eGFR**:
   - Indicates reduced kidney function. While it is an important marker, it alone does not necessitate emergency hemodialysis without life-threatening complications.
3. **Hyponatremia**:

	◦ Mildly low sodium levels are not typically an emergency indication for hemodialysis. Hyponatremia is usually managed with other treatments unless it is severe and symptomatic. 4. **Toxin Removal**: ◦ This is relevant if there is a known toxin or drug that needs to be cleared from the body. In this case, no such toxin is mentioned, so it is not the primary reason for dialysis. **Correct Option: Hyperkalemia**: • **Reason**: Hyperkalemia is a life-threatening condition that requires immediate intervention due to the risk of cardiac arrhythmias, as indicated by the tall, peaked T-waves on the ECG.
Key 66	**Quick Important Notes (Contrast-Induced Nephropathy).** • To reduce the risk of contrast-induced nephropathy in patients with chronic kidney disease, adequate **hydration** is crucial. • Administering 0.9% normal saline intravenously before and after the contrast exposure is the most effective method. • This approach helps to maintain renal perfusion and dilute the contrast agent, reducing its harmful effects on the kidneys.